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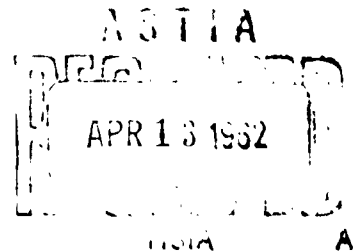
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ASD TECHNICAL REPORT 61-740

**REGULATION OF CUTANEOUS CIRCULATION  
DURING BODY HEATING****ALRICK B. HERTZMAN****SAINT LOUIS UNIVERSITY****DECEMBER 1961****CONTRACT No. AF 33(616)-7077**

**BIOMEDICAL LABORATORY  
AEROSPACE MEDICAL LABORATORY  
AERONAUTICAL SYSTEMS DIVISION  
AIR FORCE SYSTEMS COMMAND  
UNITED STATES AIR FORCE  
WRIGHT-PATTERSON AIR FORCE BASE, OHIO**

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ASD TR 61-740	<p>Saint Louis University, St. Louis, Missouri</p> <p>REGULATION OF CUTANEOUS CIRCULATION DURING BODY HEATING, by A. B. Hertzman. December 1961. 29p. incl. illus. tables. 55 refs. (Proj. 7222; Task 722204). Unclassified report</p> <p>This report summarizes current information concerning the regional adjustments of cutaneous vascular tone during body heating. Most of the data to which reference is made were obtained in the writer's laboratory. The importance of the local skin temperature in accounting for the cutaneous vasodilatation</p> <p>( over )</p>	<p>UNCLASSIFIED</p> <p>I. Hertzman, A. B. II. Aeronautical Systems Division, Aerospace Medical Laboratory, Wright-Patterson Air Force Base, Ohio III. Contract No. AF 33(616)-7077</p> <p>UNCLASSIFIED</p>	<p>ASD TR 61-740</p> <p>Saint Louis University, St. Louis, Missouri</p> <p>REGULATION OF CUTANEOUS CIRCULATION DURING BODY HEATING, by A. B. Hertzman. December 1961. 29p. incl. illus. tables. 55 refs. (Proj. 7222; Task 722204). Unclassified report</p> <p>This report summarizes current information concerning the regional adjustments of cutaneous vascular tone during body heating. Most of the data to which reference is made were obtained in the writer's laboratory. The importance of the local skin temperature in accounting for the cutaneous vasodilatation</p> <p>( over )</p>	<p>UNCLASSIFIED</p> <p>I. Hertzman, A. B. II. Aeronautical Systems Division, Aerospace Medical Laboratory, Wright-Patterson Air Force Base, Ohio III. Contract No. AF 33(616)-7077</p> <p>UNCLASSIFIED</p>	<p>ASD TR 61-740</p> <p>is emphasized and the role of bradykinin is discounted. The regional differences in the onset, temporal sequence, and extent of the cutaneous vasodilatation are not explained by reference to a central thermostat in the hypothalamus.</p>	<p>UNCLASSIFIED</p>	<p>UNCLASSIFIED</p>
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**REGULATION OF CUTANEOUS CIRCULATION  
DURING BODY HEATING**

**ALRICK B. HERTZMAN  
SAINT LOUIS UNIVERSITY**

**DECEMBER 1961**

**CONTRACT MONITOR: JOHN F. HALL, JR.  
CONTRACT No. AF 33(616)-7077  
PROJECT No. 7222  
TASK No. 722204**

**BIOMEDICAL LABORATORY  
AEROSPACE MEDICAL LABORATORY  
AERONAUTICAL SYSTEMS DIVISION  
AIR FORCE SYSTEMS COMMAND  
UNITED STATES AIR FORCE  
WRIGHT-PATTERSON AIR FORCE BASE, OHIO**

## FOREWORD

The summary herein of current information and concepts concerning adjustments in the cutaneous circulation during heat exposure was prepared in fulfillment of contract AF 33(616)-7077, Project 7222, "Biophysics in Flight," and Task 722204, "Human Thermal Stress in Extended Environment," administered by the Aerospace Medical Laboratory, Aeronautical Systems Division, Wright-Patterson Air Force Base, Ohio. Mr. John F. Hall, Jr., Chief of the Biothermal Section, Biomedical Laboratory, served as the contract monitor.

The experimental information illustrated in figures 4 and 5 was supplied by Dr. Kiyoshi Seki with the support of USPHS grant H-4939.

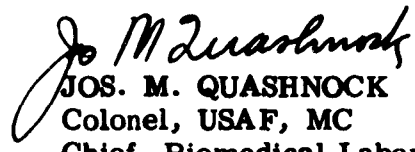
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## ABSTRACT

This report summarizes current information concerning the regional adjustments of cutaneous vascular tone during body heating. Most of the data to which reference is made were obtained in the writer's laboratory. The importance of the local skin temperature in accounting for the cutaneous vasodilatation is emphasized and the role of bradykinin is discounted. The regional differences in the onset, temporal sequence, and extent of the cutaneous vasodilatation are not explained by reference to a central thermostat in the hypothalamus.

## PUBLICATION REVIEW

  
JOS. M. QUASHNOCK  
Colonel, USAF, MC  
Chief, Biomedical Laboratory  
Aerospace Medical Laboratory

## I. INTRODUCTION

The purpose of this report is to summarize the currently available information on the regulation of the cutaneous circulation during exposure of the nude resting subject to environmental heat. Principal attention will be given to those adjustments which pertain primarily to the vascular transfer of heat to the skin. The thermoregulatory mechanisms may also elicit, elsewhere in the body, circulatory changes which do not seem necessary to this heat transfer; e.g., a decrease in the vascular resistance in the splanchnic bed.<sup>1</sup> Nevertheless, such secondary effects may be pertinent to a theory of temperature regulation.

The circulation through the skin and the upper respiratory mucosa seems to be concerned primarily with heat transfer and only secondarily with metabolic requirements. In all of the inner tissues, the need for heat removal is usually met automatically and secondarily by the vascular supply of oxygen. Thus, the oxidative heat which would result from the consumption of 1 cm<sup>3</sup> of oxygen would elicit an increase of only 0.05° C. in the temperature of the blood passing through the tissue, if this quantity of oxygen were supplied by 100 cm<sup>3</sup> of blood. The relation is obviously independent of blood flow which is controlled by the metabolic call, not by the thermal requirements. The statement does not negate direct thermal effects on the blood vessels of the deeper tissues or adjustments in them due to vasomotor discharges resulting from the activities of the thermoregulatory centers of the brain.

The vascular convection of heat to the skin is the product of the cutaneous blood flow and the change in blood temperature. Therefore, three variables may be expected to influence the heat transfer and to present the effector basis of regulation: blood flow, the temperature of the blood entering the site of the transfer, and the temperature of the blood leaving the site. In the warm or hot individual the vertical thermal gradients are greatly reduced so that blood enters the skin at temperatures near those of the core and leaves at a temperature near that of the surface. Teleology suggests that regulation would consider these two temperatures in adjusting cutaneous vascular tone and the rate of the cutaneous blood flow. Benzinger emphasized a close correlation between intracranial temperature and the level of cutaneous blood flow presumably due to an accurate adjustment of vasomotor tone but relegated the local skin temperature to subordinate importance.<sup>2</sup> His elegant experiments show a linear relation of conductance to the intracranial temperature above a threshold of 36.9° C. in a particular subject. Sweating also appeared at this level of internal temperature above which the cutaneous vasodilatation and sweating increased in a parallel manner.

When the cutaneous vascular responses and sweating are examined in the same region, the relations to body temperature are more complex than depicted in Benzinger's study. Thus, in an experiment performed in our laboratory 10 years ago <sup>3,4</sup> and summarized in Table 1, we noted that profuse sweating could be induced on the calf without cutaneous vasodilatation when only this area was exposed to the cool room while this subject was heated by an electric blanket which covered the rest of his body (excepting the head). This observation

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Table 1. Temperatures, blood flows, and sweating of exposed calf skin during heating of subject with an electric blanket. Room temperature 22.6° C.

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	Control period 1 hour	Heating period 1 hour
T <sub>s</sub>	31.0	29.6° C.
Sweat	0.0	.142 mgm/cm <sup>2</sup> /min
Blood flow (1)	22.	24. mm <sup>3</sup> /cm <sup>2</sup> /min
Local conductance (2)	.017	.021 cal/cm <sup>2</sup> /min/°C
<hr/>		
(1) From skin pulses	(2) Estimated	

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emphasized the necessity of examining the sweating and vascular responses in each principal skin region during various patterns of heat stresses. The investigation was initiated by the design of a photoelectric plethysmograph which could be applied to the skin of all regions of the body <sup>5,6</sup> and extended by the development of simple methods for the measurement of sweating at various loci <sup>7,8</sup>. The plethysmographic technic permitted continuous estimations of the rate of arterial perfusion <sup>9</sup> and recordings of changes in the blood content <sup>10</sup> in the skin under the photometer. The earlier observations clearly demonstrated the cutaneous vasodilatation elicited by a warmer environment <sup>3,11</sup> and also regional differences in cutaneous vasomotor responses to various stimuli <sup>12,13</sup>.

The following factors may be expected to influence the cutaneous vascular tone, the cutaneous volume flow of blood, and hence the vascular transfer of heat to the skin in any particular region of the body: the number, size and arrangement of the vascular channels, i.e., the vascularity of the skin; the vasomotor innervation of these vessels; the relations of the local skin temperature

and of local sweating to the local vascular tone and blood flow; and the arterial and venous pathways of the heat transfer. These influences and the additional one of dehydration which usually develops to various levels during heat exposure, are examined in the succeeding sections with particular attention given to the regional differences or similarities in the cutaneous vascular responses. However, despite the importance of evaluating the quantitative relations of the vascular responses in the different regions and of the measurement of the regional fractions of the total heat transfer by the blood stream to the entire skin surface of the body, this observational effort has not been made. Finally, a summary is presented of this laboratory's concept of the thermoregulatory control of the arterial blood flow in the skin.

## II. CUTANEOUS BLOOD FLOW DURING BODY HEATING.

### 1. Vascularity of the skin.

The upper level of cutaneous blood flow attainable by local vasodilatation is approximately the same in the trunk, arm and leg, but considerably higher in the palm, sole and head.<sup>11</sup> Therefore, major regional differences in rates of cutaneous arterial perfusion in the trunk, arm and leg during various heat loads must be due to corresponding regional differences in the adjustment of vascular tone rather than in the number of blood vessels. At normal levels of arterial pressure and during the maximal local vasodilatation elicited by vasodilator drugs applied locally, the arterial perfusion may attain a rate of  $0.2 \text{ cm}^3/\text{cm}^2 \text{ skin/minute}$  in the trunk, arm, and leg.<sup>11</sup> Rates three times as great were noted in the palm at the time of maximal vasodilatation.<sup>11</sup>

The regional fractions of the total cutaneous arterial perfusion vary greatly with ambient conditions and the metabolic rate of the individual. Thus, the blood flows in the palm, sole, and head comprise nearly one half of the total cutaneous blood flow in the comfortable resting subject <sup>11</sup> but, during conductances of  $2.3 \text{ L/M}^2/\text{minute}$ , these regional fractions would not be more than one fourth of the total. The regional fractions of the total cutaneous evaporation exhibited a similar re-distribution during body heating. <sup>14,15,16,17</sup>

The method of recording the skin pulses provides estimates of the local cutaneous blood flow which agree fairly well with calculations based on venous occlusion plethysmography. Table 2 shows such a comparison. The method of physiological skinning of the forearm by the electrophoresis of epinephrine indicated that the cutaneous blood flow was very small when the forearm blood flow was about  $15\text{-}30 \text{ mm}^3/\text{cm}^3 \text{ forearm/minute}$ . <sup>18</sup> Skin pulse data agreed. Determinations were so uncertain that they were not entered in the

Table 2. Forearm blood flows during body heating.

Forearm	Total forearm flow	Skin flow estimated from Column 2	Skin flow from skin pulse
	(a)	(b)	(c)
Cool subject	30 (1)	0.0	—
35.7	90 (2)	44	66 (3)
37.7	230 (2)	148	186 (3)
<div> <div>(a) <math>\text{mm}^3/\text{cm}^3/\text{min.}</math></div> <div>(b) <math>\text{mm}^3/\text{cm}^2/\text{min.}</math></div> </div> <div> <div>(1) Ref. 18</div> <div>(2) Ref. 19</div> <div>(3) Ref. 21,22</div> </div>			

table. From this low level, forearm blood flow increased during body heating to 230  $\text{mm}^3/\text{cm}^3$  forearm/minute when the skin and rectal temperatures were 37.5° and 38.6° C, respectively. <sup>19</sup> Accepting that no increase occurred in blood flow in the forearm muscles <sup>20</sup>, the corresponding skin blood flows were calculated, using the value of 1.35 for the ratio of surface area to volume of the forearm, and entered in the third column of table 2. These estimates may be compared with the skin pulse data at the same levels of forearm skin temperature. <sup>21,22</sup>

The physiological needs which might require high rates of blood flow in the palm, sole, and face have not been identified. Palmar and plantar flows may serve protective needs during cold exposures but the high rates during heat exposures effect no more heat transfer than would lower flow rates because the local sweat rates are not high. <sup>14,15,17</sup> However, since much of the palmar and plantar venous drainage may return to the central veins via the subcutaneous veins of the forearm and calf, further cooling of the blood is possible. A high rate of blood flow in the skin of the head and neck would not only protect the brain against environmental temperature but also tend to approximate brain and core temperatures. The result should expedite brain control over the body's heat content. The apparent absence of a powerful vasoconstrictor innervation in this region, <sup>12,13,23,24,25</sup> is of considerable interest to such speculations.

The maximum capacity for vasodilatation in the skin permits the convective transfer of metabolic heat to the skin even at the highest possible rates of oxidative heat formation without requiring impractical differences in core and surface temperatures and without large changes in perfusion pressure or in cardiac output. Thus with an oxygen consumption of  $2.5 \text{ L/M}^2/\text{minute}$  and a core-surface temperature difference of  $5^\circ \text{C}$ , the required cutaneous blood flow would be  $2.5 \text{ L/M}^2/\text{minute}$  which quantity could be supplied by maximum vasodilatation with little increase in arterial blood pressure. The deviation of blood to the skin would not be more than 15 percent of the cardiac output at this time.

## 2. Vasomotor innervation of the skin.

Recent reports indicate the presence of arterioconstrictor and arteriodilator nerve fibers in the skin of the arm and leg <sup>26</sup>, and arterioconstrictor innervation of the ear <sup>23</sup>, and an arteriodilator supply to the face <sup>23,24</sup>. The palm and sole receive only vasoconstrictor fibers <sup>27</sup>. The skin veins are supplied by constrictor fibers.

The arterioconstrictor innervation of the skin of the arm and leg does not seem to be very important. Its blockade results in only a small increase in blood flow <sup>26</sup> and that only when the subject is cold. Most of the increase during body heating has been attributed to an active vasodilatation due to arteriodilator fibers <sup>26</sup>. The increase in manual and pedal blood flows <sup>27</sup> and also that in ear blood flow <sup>23</sup> during heat exposures seems to be due to decreased arterioconstrictor tone. These statements are based on procedures which may prove to have been inadequate. Verified sympathetic ganglionic blockade as well as direct stimulation of sympathetic outflows may require quantitative revisions of current information. Such data have not been reported.

Thermal stimuli to the skin of one region may elicit reflex vasomotor responses in another region. Both cold and heat stimuli bring about vascular responses in the hand when they are applied to the skin elsewhere in the body <sup>28</sup>. Radiant heat directed on the legs results in vasodilatation in the hand even when the blood flow to the heated legs is occluded <sup>29</sup>. This response is prevented by lumbar sympathectomy. It is clear that afferent impulses elicited by changes in skin temperature, may influence cutaneous vascular tone in regions distant from that in which the change in temperature occurs, but the quantitative importance of these reflexes has not been appraised.

The additional influence of axone-type reflexes will be considered below.

### 3. Regional cutaneous vascular responses to rising ambient temperature.

Exposure of the nude resting human to a slowly increasing ambient temperature provided a suitable circumstance for the study of cutaneous vascular responses to body heating <sup>21,22</sup>. The temporal sequences in these responses as well as in regional sweating were extended thus permitting a surer identification of regional differences and their relation to suspected causes. The results of these experiments which have been presented in technical reports <sup>21,30</sup> and elsewhere <sup>22,31</sup>, clearly confirmed the implications of the earlier studies <sup>3,4,5,11</sup> in this laboratory. Regional differences in the time of onset and progress of the cutaneous vasodilatations were often observed. They seemed to be characteristic of the individual subject. Thus in some reported experiments <sup>21</sup> there was no apparent relation between the responses in the finger pad and forearm during the heat exposure. Cutaneous vasodilatation in the forearm occurred early in the heat exposure while the finger vessels constricted progressively at the same time, finally dilating late in the heat exposure. In some experiments, cutaneous vasodilatations occurred simultaneously and developed similarly in the finger and forearm. The increase in blood flow might continue during the entire period of rising ambient temperature or stabilize at some plateau value.

Observations of the cutaneous vascular responses were also made on the toe, calf, thigh, ear, cheek, and forehead as well as on the finger and forearm. Some of these observations have been recorded <sup>30,31</sup>. The individual differences in these regional vascular responses were as striking as those which had been observed in regional sweating <sup>32</sup>. These were partially illustrated in previous experiments <sup>30</sup> on three subjects who differed in their acceptance of the heat stress and also in the cutaneous vasodilatations. One subject apparently was heat acclimatized. Sweating began earlier on him than on the other two subjects exposed to essentially the same heat stress. The cutaneous vasodilatation was slight in the calf of this subject and even absent in other experiments on him. At the same time, blood flow increased to a high level in his forearm skin and then stabilized at this level as forearm sweating became marked. A second subject, apparently intermediate between the other two subjects in the time of onset of sweating, exhibited early vasodilatation in the toe and calf skin which soon stabilized at a relatively low level although that in the forearm skin continued to increase throughout the rise in ambient temperature. The third subject showed marked vasodilatation in both the calf and forearm. He seemed to be a "poor" sweater. Forearm sweating began an hour later than that in the first subject exposed to comparable changes in ambient temperature. The relations between the skin pulses in the forearm and calf in these

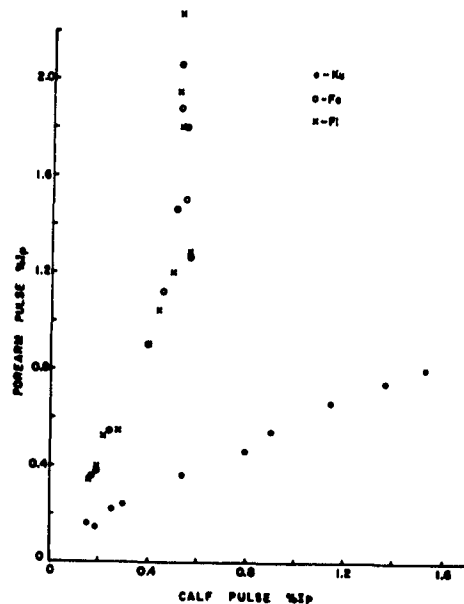


Figure 1. Relation of skin pulses in calf and forearm during heating of subject by rising chamber temperature. Three different experiments on three different subjects: solid circles, subject Ku; open circles, subject Fe, crosses, subject Fl. Amplitudes of photoelectrically recorded skin pulses in percent decrement in photoelectric current (%Ip).

three subjects are shown in Figure 1. The amplitudes of the skin pulses are expressed as percent decrement in the photoelectric current (%Ip). In all three subjects, the cutaneous vasodilatation increased proportionally in the calf and forearm during the early part of the body heating. However, in subjects Fe and Fl, the cutaneous vasodilatation in the calf was only about one third as great as that in the forearm. This difference was not due to corresponding differences in the cutaneous vascularity in the two regions - maximal blood flows at any given level of perfusion pressure are about the same in these two skin regions.<sup>11</sup> In subject Ku, the cutaneous vasodilatation in the calf was about twice as great as that in the forearm throughout the entire period of body heating. The linear relation of the skin pulses in the two regions may have been due to the way in which the vasomotor controls influenced the vascular tone. The higher temperatures in the skin of subject Ku may have been relevant to the greater vasodilatation in his calf skin.



It seemed improbable that these regional differences in the cutaneous vascular responses could be accounted for simply by a rising hypothalamic temperature. Neither did it appear probable that normal individuals would differ greatly from each other respecting the sympathetic innervations of the skin. Three possible influences on the local vascular tone seemed to deserve study in an attempt to explain the local vascular responses to the heat stress imposed on the subject. Two of these, namely the relation of the local skin temperature to the local blood flow and the relation of local sweating to the local blood flow, are considered in the following sections. The third possibility, that of selectivity in the regional distribution of the vasomotor discharges <sup>12</sup> has not been studied critically in relation to the regional thermoregulatory responses of the cutaneous circulation.

#### 4. Local skin temperature and local skin blood flow.

The direct effects of temperature on vascular smooth muscle are documented in many papers dealing with the responses of blood vessels to heat, cold, diathermy and infrared radiation. The importance of these effects was given considerable attention in the analysis of the cutaneous vascular responses to body heating in our studies <sup>3,4,21,22,30,31</sup>. Thus, in several experiments, it was noted that the skin temperature of the calf was consistently less than that of the forearm during the entire heat exposure and correspondingly, that the cutaneous vasodilatation was less in the calf <sup>30,31</sup>. If the local skin temperature controlled the local vascular tone, as such experiences suggested, it seemed possible to inhibit the cutaneous vasodilatation in a small area during the exposure of the subject to a slowly rising ambient temperature, simply by keeping this area cool during the body heating. The results of such experiments have been recorded <sup>21,22</sup>. The thermode which was designed to test this possibility prevented the vasodilatation in the area covered by the thermode until this area's temperature was allowed to approach that of the other forearm which was wholly exposed to the ambient temperature. The relations between the local skin temperature and the local skin pulses in such experiments are illustrated in Figure 2 which was prepared from two experiments on the same subject. The fit of the observations on each forearm to the curvilinear relation between skin temperature and skin pulse appeared to be unaffected by the thermode. The skin temperatures of the right forearm (exposed) were higher than those of the area of the left forearm under the thermode. Likewise, the skin pulses were greater in the right forearm. In similar unreported experiments, in which a specially designed stream calorimeter, incorporating a photoelectric plethysmograph, was used to control the local skin temperature, both the conductance (as calculated from the measured heat transfer) and the skin pulses correlated with the local skin temperature during the heating of the subject with an electric blanket <sup>33</sup>.

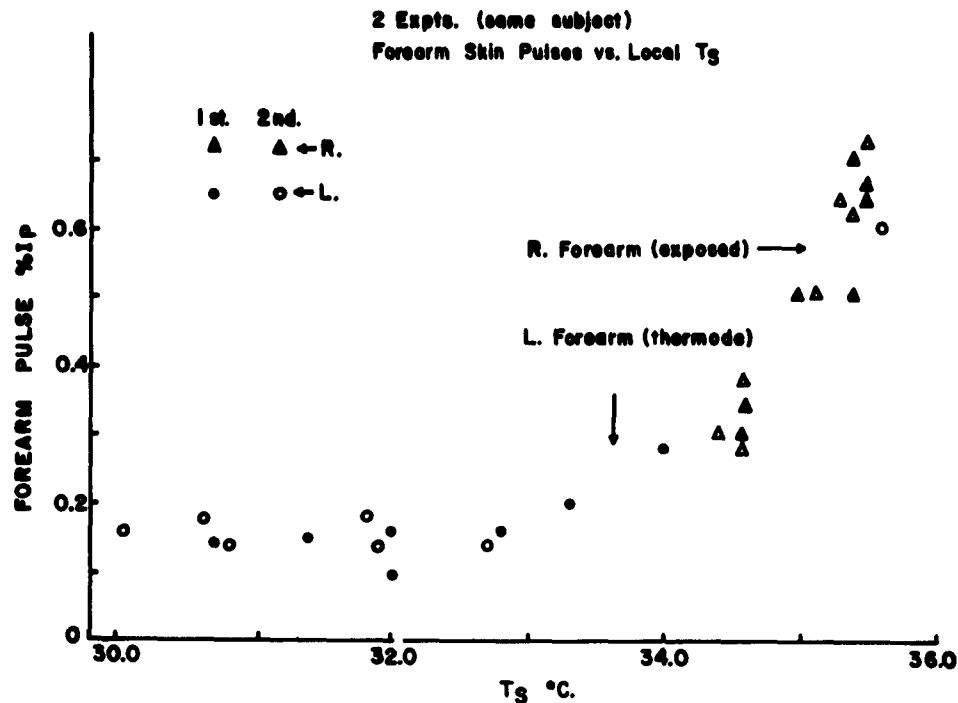


Figure 2. Local skin temperatures and local skin pulses in right (triangles) and left (circles) forearms, during rising chamber temperature, while a small area ( $24 \text{ cm}^2$ ) of the left forearm was covered by a thermode which kept the skin temperature of that area at the indicated values. Both forearms were exposed to the chamber climate, except for the area on the left forearm covered by the thermode. The results of two experiments on the same subject are shown, those of the first experiment by solid circles and triangles, those of the second experiment by open circles and triangles.

In contrast to the inhibition of the vascular dilatation under the thermode, sweating under the thermode as well as distal to it was not measurably affected by the thermode. Sweating began and increased in these areas as on the control forearm even though vasodilatation did not occur under the thermode as long as it was kept cool. The evidence seemed decisive that within the limits of this particular experiment, local skin temperature dominantly controlled the local circulation without influencing the local thermoregulatory sweating.

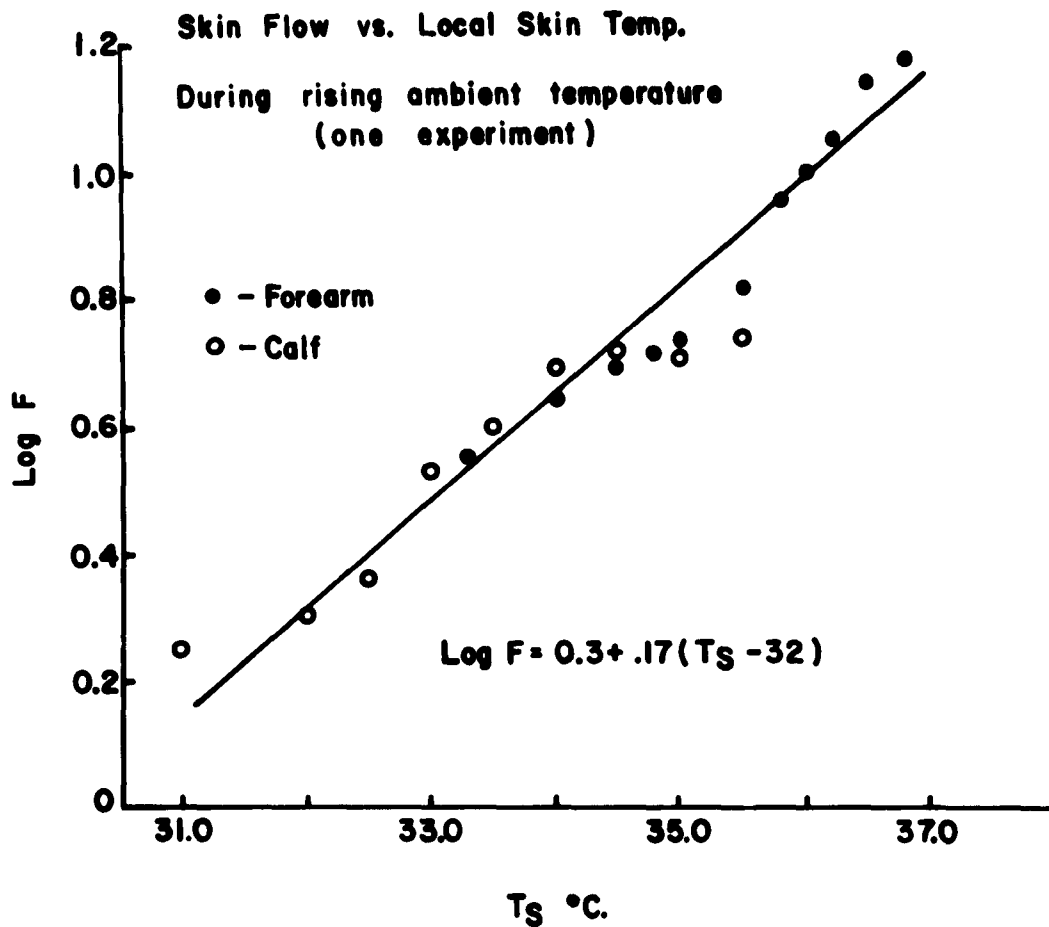


Figure 3. Relation of local skin temperature to the logarithm of the local skin blood flow. The amplitude of the skin pulse was converted to equivalent blood flow (cc/100 cm<sup>2</sup>/min). Data from one experiment.

How does the local skin temperature influence the local vascular tone? Freeman showed that in the sympathectomized hand, the relation of blood flow to hand temperature was logarithmic<sup>34</sup>, indicating a temperature dependent rate which fitted the Arrhenius equation. The more recent measurements by Peacock<sup>35</sup> of blood flow in a normal hand immersed in a bath of various temperatures may be expressed by the equation

$$\text{Log } F = 0.3 + .06 (T_{\text{bath}} - 22)$$

where  $F$  = hand blood flow in  $\text{cm}^3/100 \text{ cm}^2/\text{min}$ .

A logarithmic relation of the skin pulses to the local skin temperature was apparent also in some of our experiments. This is shown in Figure 3 where previous experimental data<sup>30</sup> is treated by charting the logarithm of the cutaneous blood flow (calculated from the flow equivalence of the skin pulse) in the forearm and calf against the local skin temperature. The equation for the line is

$$\text{Log } F = 0.3 + 0.17 (T_s - 32)$$

where  $F$  is the cutaneous blood flow in  $\text{cc}/100 \text{ cm}^2/\text{minute}$  and  $T_s$  is the local skin temperature in  $^{\circ}\text{C}$ . The deviations of the individual points from the regression equation were probably real and less due to observational error than to other influences on the vessels. Although the points for the calf and forearm skins happen to fit the same equation in this particular experiment, it would be astonishing if a single temperature dependent rate were the only determinant of cutaneous vascular tone in a particular region during heat exposure of the whole body. Nevertheless, similar logarithmic plots were obtained from the data of other experiments on other subjects. This experience seems to justify our emphasis on the dominant importance of local skin temperature to the local vascular tone.

The relations of local vascular tone to local skin temperature involve neural mechanisms as well as a possibly direct thermal effect on the smooth muscle. Thus, during infrared heating of a limited area of forearm, cutaneous vasodilatation occurred in the radiated area and also distal to it, but not in the other arm<sup>36</sup>. Cutaneous nerve blocks above the heated area did not inhibit the vasodilatation, but such blocks immediately distal to the irradiated area prevented the distal extension of the vasodilatation. This evidence points to axone reflexes of considerable pertinence to the local thermal effects. Their relation to the thermode experiments was not determined.

##### 5. Local sweating and local skin blood flow.

The equation for convective heat transfer indicates no need for a precise relation between sweating and cutaneous blood flow. The cooling effect of the evaporation of sweat diminishes the requirement for blood flow by increasing the core-skin temperature difference

more than what would be true in the absence of evaporation. It would be surprising therefore to note a systematic rate of increase in cutaneous blood flow with the onset of and increment in sweating. However, since higher body temperatures are usually accompanied by cutaneous vasodilatation and sweating, these two events in the skin may appear to be related causally when the connection may in fact be coincidental. A somewhat different position has been taken by some investigators <sup>37,38</sup> who have accepted the liberation of bradykinin by the sweat gland during its secretory activity <sup>39</sup> as an important vasodilator mechanism.

If the activity of the sweat gland accounts for the cutaneous vasodilatation during body heating, the increase in blood flow should occur whenever sweating takes place. The data in Table 1 show that this need not be the case. This experiment demonstrated that fairly profuse thermoregulatory sweating could occur in a particular area without noticeable vasodilatation. No systematic temporal relations were observed between these two responses in our experiments <sup>21,30</sup>. Thus, sweating in the forearm began long after the onset of the vasodilatation in this region in some subjects while in others in whom the relation between sweating and cutaneous blood flow was approximately linear in the forearm, the sweating calf showed little vasodilatation. In some experiments, the cutaneous vasodilatation attained a plateau level while ambient temperature and sweating were still increasing. Skin temperatures correspondingly stabilized suggesting that their level was determining the vascular tone. These relations might even suggest an inhibitory influence of sweating on the progress of the vasodilatation in the sense that evaporation prevented further rise in the local skin temperature.

The bradykinin theory encounters additional difficulties in the changes in peripheral blood flow and in sweating resulting from acclimatization to heat. At the same levels of body temperature sweating is greater <sup>40</sup> and peripheral blood flow is less <sup>41</sup>, so that the ratio of sweat to blood flow is considerably greater than in the nonacclimatized subject. If the cutaneous vasodilatation were due principally to bradykinin, the reverse should be true.

One might infer from the anatomy of the cutaneous circulation <sup>40</sup> that the major fraction of the cutaneous blood flow during body heating is concerned with heat delivery rather than the energy requirements of the glands. The rapid rise in the ratio of oxygen to reduced hemoglobin in superficial venous blood during the cutaneous vasodilatation elicited by heat <sup>42</sup> supports this concept. However, direct fractionations of glandular and nonglandular blood flows in the skin are not available. The ratio of blood flow to sweating in a particular region varies widely during various heat exposures and the actions of drugs which excite the sweat glands. Thus, at the height of the influence of Methacholine, about 200 or more cm<sup>3</sup> of blood flowed through the skin during the formation of 1 cm<sup>3</sup> of sweat (personal observation). This quantity should not be required for

the supply of either water or oxygen. (These ratios are similar in magnitude to those in the kidney.) The liberation of bradykinin by the sweat gland might increase the gland's blood supply without affecting blood flows in other skin vessels. This increase could be too small a fraction of the total quantity to be detectable by present methods of measuring blood flows. The relation of plasma kinin to vascular tone is discussed by Lewis <sup>43</sup> to whom the reader is referred for additional details.

### III. VASCULAR PATHWAYS FOR HEAT TRANSFER TO THE SKIN

The regulation of the vascular convection of heat to the skin may be directed on the arterial flow to the skin and also on the venous drainage. The preceding discussion has been concerned with the cutaneous arterial responses to heat. Their relation to heat transfer was implied but not examined. The cutaneous venous responses importantly influence the blood content of the skin, the changes in temperature of the blood passing through the peripheral arteries and skin, the temperature gradients in the insulating "shell" and hence the heat transfers.

#### 1. Skin pulses and heat transfer in the skin.

A direct comparison of local cutaneous heat transfer as measured by a modified Hensel stream calorimeter <sup>44</sup> with the amplitudes of the local skin pulses is being undertaken in this laboratory. Figure 4 (offered through the courtesy of Dr. Seki) shows the simultaneous measurements of heat transfer and of skin pulses in the forearm following the local administration of Methacholine by electrophoresis. The temporal sequence of the vasodilatation in this experiment was similar to recent measurements of forearm blood flow by the venous occlusion method during the action of this drug <sup>45</sup>. Excepting for a brief discrepancy due to the thermal inertia of the skin and calorimeter immediately following the reapplication of the instrument, the skin pulses were linearly related to the rate of heat transfer. This proved to be true in both the palm and forearm during vasomotor reflexes and at various levels of blood flow as indicated by both methods. However, in all observations, the temporal sequences were shown more precisely in the records of the skin pulses, particularly when the vascular changes were rapid or of brief duration.

A direct calibration of the relationship between the volume pulse and the rate of arterial perfusion in a particular skin region has not been available in regions other than the fingers. Neither has the actual vascular site of the cutaneous volume pulse been determined. However, the presence of pressure pulsations in small veins of the rabbit's ear and their reduction during stimulation of the sympathetic supply <sup>46</sup> indicates that part of the volume pulsation may occur on the venous side of the capillary bed. A decrease

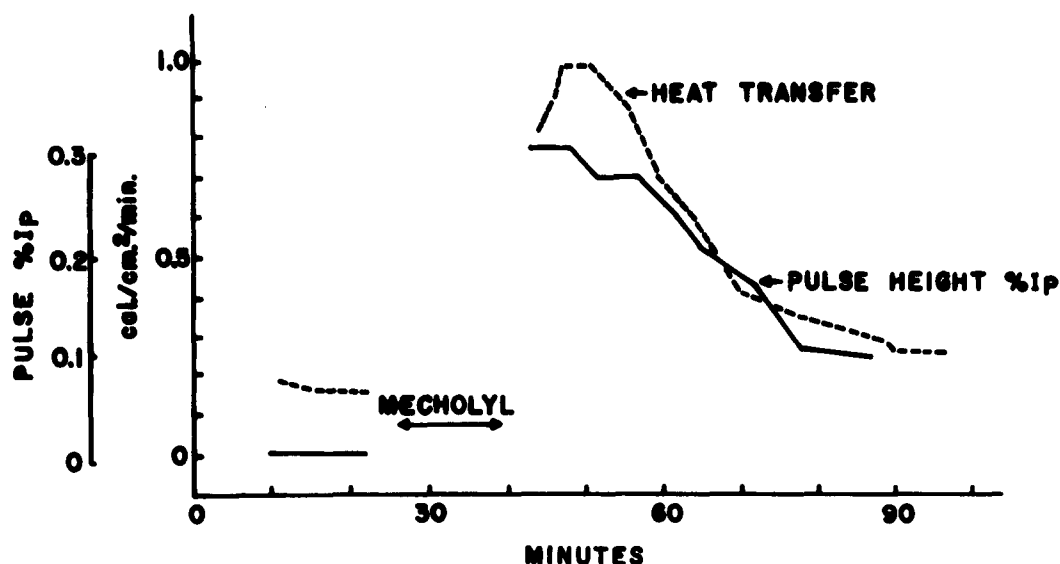


Figure 4. Simultaneous measurements of the skin pulses and heat transfer to a specially designed stream calorimeter before and following the application of Methacholine by electrophoresis to the forearm. The design of the instrument permitted the measurements to be made on the same skin area. (Courtesy of Dr. Seki of this laboratory).

in the amplitude of the pressure pulses and in pulse pressure occurred in the small arteries of the dog's paw when the lumbar sympathetic chain was stimulated<sup>47</sup>. Such data suggest a general correlation between the volume pulsation and the local vascular resistance and hence the blood flow. In recent unreported experiments by Dr. Seki in this laboratory, the relations between conductances and skin pulses have been examined in the same area with a specially designed stream calorimeter in which a photoelectric plethysmograph was incorporated. The details of these studies will be reported later in a separate communication. They demonstrated a linear relation between conductance and the skin pulse in forearm and calf skin during the action of vasoactive substances and during body heating. These experiments further confirmed the validity of using the skin pulses as a direct measure of the rate of arterial perfusion of the skin.

## 2. Cutaneous venous responses and heat transfer.

Distension of the subcutaneous veins is commonly observed in the hot subject. A large fraction of the venous congestion must be due to the increased arterial perfusion of the skin but part is

probably accounted for by decreased venomotor tone. The evidence respecting venomotor responses has been reviewed elsewhere <sup>27</sup>. The practical difficulty of distinguishing the simultaneous occurrence of "active" and "passive" venous hyperemia is illustrated in figure 5, a plot of simultaneous records of the volume pulses of the finger pad, plethysmogram of the finger pad, and plethysmogram of a subcutaneous vein on the dorsum of the hand. The three records were taken by means of photoelectric plethysmographs. In this hot subject, the vasoconstrictor reflex elicited precisely simultaneous decreases in the three quantities being recorded, suggesting a venomotor as well as an arteriomotor response. Recovery in the skin pulse record was swift while both plethysmograms required at least a minute after the minimum for complete recovery. In some responses, the venous recovery proceeded in two stages, the first running parallel to that in the volume pulses, the second at a slower rate. Distinction between venous filling due to the arterial inflow and that resulting from relaxation of the venous muscle did not seem possible. In reactions in forearm skin, the photoelectric plethysmograms often showed decreases in blood content without a change in the skin pulse when vasoconstrictor reflexes were elicited. Similar data were obtained with plethysmographs enclosing the whole forearm <sup>48</sup>. It is clear that venomotor controls influence the cutaneous venous system but their importance to the body's responses to heat has not been evaluated.

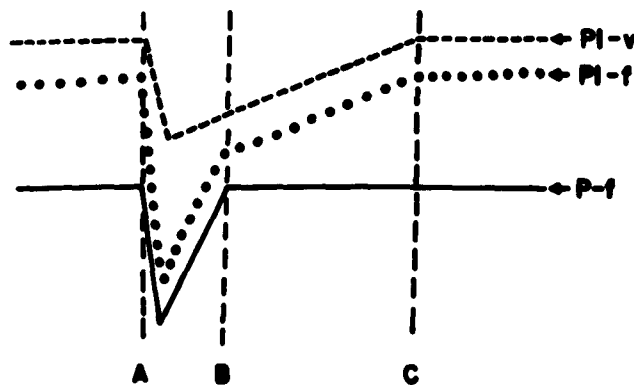


Figure 5. Simultaneous records of the volume changes of a subcutaneous vein on the dorsum of the hand (Pl-v) and of the finger pad (Pl-f) and of the latter's skin pulses (P-f) during and following a vasoconstrictor reflex. All records were taken by specially designed photoelectric plethysmographs and were charted in mms of excursion of the record. (Courtesy of Dr. Seki of this laboratory).



Measurements of intravascular temperatures showed that heat exchanges must occur between the venae comitantes and the arteries which they accompany <sup>49</sup>. This arrangement of thermal counter-currents should be particularly effective in lowering heat losses from the extremities during exposure to cold. It seems to be used by those animals which are exposed to cold air or cold water <sup>50</sup>. When the body warms because of a hot environment or due to higher heat production, the effectiveness of the thermal counter-current would diminish because of a higher blood flow in the artery and higher temperatures in the returning venous blood, irrespective of any simultaneous venomotor responses which could be expected to favor the diversion of blood into the cutaneous veins. These implied relations require quantitative studies which do not seem to be available in the literature.

The direct vascular convection of heat from contracting muscle to overlying skin has been demonstrated <sup>51</sup>. Small venous connections between the cutaneous and muscular venous systems are employed for this purpose. The advantages from the viewpoint of circulatory economy are obvious. They are indicated by calculations of the heat transfer which may become a sizeable fraction of the heat production of the active muscle.

#### IV. DEHYDRATION

Several frequently encountered disturbances of body functions, such as fever, peripheral vascular disease, sympathectomy, autonomic blockade in the treatment of hypertension, etc., may be expected to influence the regulation of the cutaneous circulation and its relations to body temperatures. Their discussion is beyond the scope of this paper. However, a brief reference to the effects of dehydration is appropriate because this always occurs to a varying degree during sweating.

The rise in body temperature during dehydration accompanying heat exposure was attributed by Adolph et al <sup>52</sup> to a failure of the peripheral circulation. Reasons have been given recently <sup>53,54</sup> for assigning to a rising threshold of sweating the principal role in the heat storage and for discounting peripheral circulatory failure as directly related to the increase in body temperature. In fact, there was no evidence in this study that the cutaneous blood flow had decreased sufficiently to prevent an adequate vascular convection of heat. However, the failure of the cutaneous blood flow to increase as the body temperatures rose indicated that the vasodilating effect of heat was being offset by a vasoconstrictor influence which was not identified. It seemed particularly significant that the rise in skin and core temperatures failed to elicit further cutaneous vasodilatation and sweating as

would have been true in the normally hydrated subject exposed to the same ambient temperature. As has been suggested elsewhere 27, the information supplied by the thermo-receptors, the osmo-receptors and the volume receptors may be mutually contradictory and correspondingly modify the influences of the sudomotor and vasomotor systems.

#### V. THE THERMOREGULATORY CONTROL OF THE ARTERIAL BLOOD FLOW IN THE SKIN

Emphasis was placed in our recent papers 21,22,30,31 on multiple factors in the thermoregulatory control of the cutaneous circulation. To the authors, the analogies in the regulation of breathing and of body temperature suggested that both peripheral and central influences would affect the control of the cutaneous vascular system and the transfer of heat into it. The local skin temperature and the liberation of bradykinin by the sweat gland seem to be significant peripheral influences, but of these two the former appears to be far more powerful as far as the total cutaneous perfusion is concerned. The effect of central temperature on the vasomotor control of the cutaneous blood vessels is often obscured by other influences acting on them.

Currently available information supports the following position:

1. The vasomotor influences of the hypothalamus facilitate the outflow from the intermediolateral cell column of the spinal cord, of either vasodilator or vasoconstrictor impulses to the skin. The descending impulses to the cord are probably nondiscriminatory with respect to the regional vasomotor responses, i.e., the hypothalamus does not show "local sign." The decrease in splanchnic resistance during body heating <sup>1</sup> would be difficult to explain on any other basis. Regional differences in the cutaneous vasomotor responses would be due to the superposition of other effects, e.g., afferent impulses acting on the spinal vasomotor neurones, local temperature, etc.

2. The regional differences in the cutaneous vascular responses to body heating in a particular experiment may be due to:

- a) Spinal cord reflexes elicited by thermo-sensory receptors in the periphery (skin).
- b) Direct thermal effects on the blood vessels.
- c) Axone reflexes resulting from thermal stimulation of the skin.

These separate influences act at various levels of quantitative significance in various types of heat load. The peripheral influences are not minor. They may dominate a particular situation.

The entrance of complicating factors in this schema is illustrated by the differences in the levels of the cutaneous blood flow in two common experiences. During exercise the conductances estimated at the same levels of skin and rectal temperatures are considerably higher than in the resting subject <sup>55</sup>. This might represent a downward movement of the thermal threshold of the cutaneous vasodilator nerve mechanisms. An analogous phenomenon is seen in the respiratory responses to carbon dioxide in the resting and exercising subject. Heat acclimatization presents an apparently different situation. The circulatory responses, including cutaneous blood flow, to heat are decreased while sweating is greater when comparisons are made at the same levels of body temperatures. The regulations of sweating and of cutaneous blood flows almost appear to be dissociated.

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